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L6	L5 and l4	216	L6
L5	dennis.in.	6591	L5
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L3	L2 and peptide	64212	L3
L2	(factor VII binding) and (Factor VIIa)	339914	L2
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Search Results - Record(s) 1 through 10 of 216 returned.

☐ 1. Document ID: US 20060154358 A1

L6: Entry 1 of 216 File: PGPB Jul 13, 2006

PGPUB-DOCUMENT-NUMBER: 20060154358
PGPUB-FILING-TYPE:
DOCUMENT-IDENTIFIER: US 20060154358 A1

TITLE: Systems and methods for ex-vivo organ care

PUBLICATION-DATE: July 13, 2006

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Hassanein; Waleed	Andover	MA	US
Bringham; Richard	North Andover	MA	US
Cecere; Giovanni	Sudbury	MA	US
Elbetanony; Ahmed	North Andover	MA	US
Fishman; Robert	Somerville	MA	US
Goff; Larry	Andover	MA	US
Khayal; Tamer	North Andover	MA	US
Kyi; Stanley	Andover	MA	US
Newell; Scott	Ipswich	MA	US
Ochs; Burt	Andover	MA	US
Sousa; Dennis	Stoughton	MA	US
Taylor; Ronald	Chester	NH	US
Rourke; Jonathan	Belmont	MA	US
Algamil; Hossam	Scranton	PA	US
Carpenter; David	Jaffrey	NH	US
Havner; Robert	Lynnfield	MA	US
Menn; Dmitri	Marblehead	MA	US

US-CL-CURRENT: 435/284.1; 435/286.5

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

☐ 2. Document ID: US 20060154357 A1

L6: Entry 2 of 216 File: PGPB Jul 13, 2006

PGPUB-DOCUMENT-NUMBER: 20060154357
PGPUB-FILING-TYPE:
DOCUMENT-IDENTIFIER: US 20060154357 A1

TITLE: Systems and methods for ex-vivo organ care

PUBLICATION-DATE: July 13, 2006

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Hassanein; Waleed	Andover	MA	US
Bringham; Richard	North Andover	MA	US
Cecere; Giovanni	Sudbury	MA	US
Elbetanony; Ahmed	North Andover	MA	US
Fishman; Robert	Somerville	MA	US
Goff; Larry	Andover	MA	US
Khayal; Tamer	North Andover	MA	US
Kyi; Stanley	Andover	MA	US
Newell; Scott	Ipswich	MA	US
Ochs; Burt	Andover	MA	US
Sousa; Dennis	Stoughton	MA	US
Taylor; Ronald	Chester	NH	US
Rourke; Jonathan	Belmont	MA	US
Algamil; Hossam	Scranton	PA	US
Carpenter; David	Jaffrey	NH	US
Havner; Robert	Lynnfield	MA	US
Menn; Dmitri	Marblehead	MA	US

US-CL-CURRENT: 435/284.1; 435/286.5

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

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☐ 3. Document ID: US 20060153832 A1

L6: Entry 3 of 216 File: PGPB Jul 13, 2006

PGPUB-DOCUMENT-NUMBER: 20060153832
PGPUB-FILING-TYPE:
DOCUMENT-IDENTIFIER: US 20060153832 A1

TITLE: Immunomodulating polymers

PUBLICATION-DATE: July 13, 2006

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Tzianabos; Arthur O.	Reading	MA	US
Kasper; Dennis L.	Charlestown	MA	US
Onderdonk; Andrew B.	Westwood	MA	US
Wang; Ying	Brookline	MA	US

US-CL-CURRENT: 424/131.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

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☐ 4. Document ID: US 20060134104 A1

L6: Entry 4 of 216 File: PGPB Jun 22, 2006

PGPUB-DOCUMENT-NUMBER: 20060134104
PGPUB-FILING-TYPE:
DOCUMENT-IDENTIFIER: US 20060134104 A1

TITLE: Humanized anti-cmet antagonists

PUBLICATION-DATE: June 22, 2006

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Dennis; Mark S.	San Carlos	CA	US
Billeci; Karen	San Bruno	CA	US
Young; Judy	San Carlos	CA	US
Zheng; Zhong	Foster City	CA	US

US-CL-CURRENT: 424/133.1; 424/146.1, 530/388.26

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

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☐ 5. Document ID: US 20060127360 A1

L6: Entry 5 of 216 File: PGPB Jun 15, 2006

PGPUB-DOCUMENT-NUMBER: 20060127360
PGPUB-FILING-TYPE:
DOCUMENT-IDENTIFIER: US 20060127360 A1

TITLE: Multi-antigen vectors of melanoma

PUBLICATION-DATE: June 15, 2006

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Berinstein; Neil	Toronto	CA	CA
Tartaglia; Jim	Aurora	MA	US
Parrington; Mark	Bradford	MA	CA
Panicali; Dennis L.	Cambridge		US
Gritz; Linda	Somerville		US

US-CL-CURRENT: 424/93.2; 435/456

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

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☐ 6. Document ID: US 20060122377 A1

L6: Entry 6 of 216 File: PGPB Jun 8, 2006

PGPUB-DOCUMENT-NUMBER: 20060122377
PGPUB-FILING-TYPE:
DOCUMENT-IDENTIFIER: US 20060122377 A1

TITLE: CDR-repaired antibodies

PUBLICATION-DATE: June 8, 2006

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Dennis; Mark S.	San Carlos	CA	US

US-CL-CURRENT: 530/387.3

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

☐ 7. Document ID: US 20060121550 A1

L6: Entry 7 of 216 File: PGPB Jun 8, 2006

PGPUB-DOCUMENT-NUMBER: 20060121550
PGPUB-FILING-TYPE:
DOCUMENT-IDENTIFIER: US 20060121550 A1

TITLE: Modulation of PDE11A activity

PUBLICATION-DATE: June 8, 2006

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Burslem; Martyn Frank	Sandwich		GB
Harrow; Ian Dennis	Sandwich		GB
Lanfear; Jeremy	Sandwich		GB
Phillips; Stephen Charles	Sandwich		GB
Wayman; Christopher Peter	Sandwich		GB

US-CL-CURRENT: 435/21; 514/252.16, 514/262.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

☐ 8. Document ID: US 20060117406 A1

L6: Entry 8 of 216 File: PGPB Jun 1, 2006

PGPUB-DOCUMENT-NUMBER: 20060117406
PGPUB-FILING-TYPE:
DOCUMENT-IDENTIFIER: US 20060117406 A1

TITLE: SOYBEAN CULTIVAR 917013

PUBLICATION-DATE: June 1, 2006

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Schultze; Dennis L.	Olivia	MN	US

US-CL-CURRENT: 800/278; 435/415, 435/468, 800/312

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

☐ 9. Document ID: US 20060117405 A1

L6: Entry 9 of 216 File: PGPB Jun 1, 2006

PGPUB-DOCUMENT-NUMBER: 20060117405
PGPUB-FILING-TYPE:
DOCUMENT-IDENTIFIER: US 20060117405 A1

TITLE: Soybean cultivar 900213

PUBLICATION-DATE: June 1, 2006

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Schultze; Dennis L.	Olivia	MN	US

US-CL-CURRENT: 800/278; 435/415, 435/468, 800/312

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

☐ 10. Document ID: US 20060117404 A1

L6: Entry 10 of 216 File: PGPB Jun 1, 2006

PGPUB-DOCUMENT-NUMBER: 20060117404
PGPUB-FILING-TYPE:
DOCUMENT-IDENTIFIER: US 20060117404 A1

TITLE: Soybean cultivar 1000126

PUBLICATION-DATE: June 1, 2006

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY
Schultze; Dennis L.	Olivia	MN	US

US-CL-CURRENT: 800/278; 435/415, 435/468, 800/312

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw Desc | Ima

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 NEWS  7  MAY 19   Derwent World Patents Index to be reloaded and enhanced
 NEWS  8  MAY 30   IPC 8 Rolled-up Core codes added to CA/CAplus and
                   USPATFULL/USPAT2
 NEWS  9  MAY 30   The F-Term thesaurus is now available in CA/CAplus
 NEWS 10  JUN 02   The first reclassification of IPC codes now complete in
                   INPADOC
 NEWS 11  JUN 26   TULSA/TULSA2 reloaded and enhanced with new search and
                   and display fields
 NEWS 12  JUN 28   Price changes in full-text patent databases EPFULL and PCTFULL
 NEWS 13  JUl 11   CHEMSAFE reloaded and enhanced
 NEWS 14  JUl 14   FSTA enhanced with Japanese patents
 NEWS 15  JUl 19   Coverage of Research Disclosure reinstated in DWPI

 NEWS EXPRESS  JUNE 30 CURRENT WINDOWS VERSION IS V8.01b, CURRENT
               MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
               AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.

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COST IN U.S. DOLLARS                                    SINCE FILE      TOTAL
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FILE 'MEDLINE' ENTERED AT 10:50:25 ON 24 JUL 2006

FILE 'BIOSIS' ENTERED AT 10:50:25 ON 24 JUL 2006
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=> s peptide and binding and factor Vii
L1          135 PEPTIDE AND BINDING AND FACTOR VII

=> s l1 and factor VIIa
L2           60 L1 AND FACTOR VIIA

=> e dennis/au
E1            1     DENNINSTON J C/AU
E2            1     DENNION G R/AU
E3            0 --> DENNIS/AU
E4           27     DENNIS A/AU
E5           14     DENNIS A B/AU
E6            8     DENNIS A C/AU
E7            2     DENNIS A D/AU
E8            2     DENNIS A E/AU
E9            2     DENNIS A F/AU
E10          36     DENNIS A J/AU
E11           7     DENNIS A J JR/AU
E12           9     DENNIS A L/AU

=> e dennis, m/au
E1            1     DENNIS Y M L/AU
E2            1     DENNIS Z G/AU
E3            0 --> DENNIS, M/AU
E4            1     DENNISEN J F/AU
E5            1     DENNISEN JON F/AU
E6            1     DENNISENKO MIKHAIL/AU
E7            7     DENNISH G/AU
E8            1     DENNISH G 3RD/AU
E9           18     DENNISH G W/AU
E10           2     DENNISH G W 3RD/AU
E11           2     DENNISH G W III/AU
E12           4     DENNISH GEORGE/AU

=> d his

     (FILE 'HOME' ENTERED AT 10:49:24 ON 24 JUL 2006)

     FILE 'MEDLINE, BIOSIS' ENTERED AT 10:50:25 ON 24 JUL 2006
L1          135 S PEPTIDE AND BINDING AND FACTOR VII
L2           60 S L1 AND FACTOR VIIA
                E DENNIS/AU
                E DENNIS, M/AU

=> d l2 ti abs ibib 1-10

L2   ANSWER 1 OF 60     MEDLINE on STN
TI   Factor VIIa stimulates endothelin-1 synthesis in
     TNF-primed endothelial cells by activation of protease-activated receptor
     2.
AB   The mechanisms linking prothrombotic changes to endothelial dysfunction
     and accelerated atheroma formation have yet to be fully defined.
     Expression of TF (tissue factor) on the endothelium is potentially an
```

initiating event as binding and activation of FVII (factor VII) can result in thrombosis. Although PAR2 (protease-activated receptor-2) is expressed on vascular endothelium, its precise physiological significance and mechanism of activation have yet to be defined. In the present study, we investigated whether PAR2 can be activated by FVIIa (activated FVII) and induce ET-1 (endothelin-1) synthesis. In bovine aortic endothelial cells pretreated with TNF (tumour necrosis factor-alpha) to increase TF expression, FVIIa stimulated ET-1 synthesis via activation of PAR2. Although FX (factor X) alone was inactive, this response was enhanced by using FVII and FX in combination. Inhibition of the proteolytic activity of FVIIa abolished the response. The PAR2 agonist peptide SLIGKV also enhanced ET-1 release on TNF-pretreated cells. The response to FVIIa was inhibited by a PAR2 antagonist peptide FSLLRY. Inhibition of the p38 MAPK (mitogen-activated protein kinase) reduced PAR2 expression and the ET-1 response. In summary, FVIIa can stimulate ET-1 synthesis in endothelial cells by activating PAR2, demonstrating a potential link between thrombotic processes and endothelial cell dysfunction.

ACCESSION NUMBER: 2005081256 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15548135
TITLE: Factor VIIa stimulates endothelin-1 synthesis in TNF-primed endothelial cells by activation of protease-activated receptor 2.
AUTHOR: Sethi Amarjit S; Lees Delphine M; Douthwaite Julie A; Corder Roger
CORPORATE SOURCE: Department of Experimental Therapeutics, William Harvey Research Institute, Barts & the London School of Medicine & Dentistry, Charterhouse Square, London EC1M 6BQ, UK.. amarjit.sethi@eht.nhs.uk
SOURCE: Clinical science (London, England : 1979), (2005 Mar) Vol. 108, No. 3, pp. 255-63.
Journal code: 7905731. ISSN: 0143-5221.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200504
ENTRY DATE: Entered STN: 16 Feb 2005
Last Updated on STN: 13 Apr 2005
Entered Medline: 12 Apr 2005

L2 ANSWER 2 OF 60 MEDLINE on STN
TI Inhibitors of Tissue Factor.Factor VIIa for anticoagulant therapy.
AB Factor VIIa (FVIIa) is a key serine protease involved in the initiation of the coagulation cascade. It is a glycosylated disulfide-linked heterodimer comprised of an amino-terminal gamma-carboxyglutamic acid-rich (Gla) domain and two epidermal growth factor (EGF)-like domains in the light chain, and a chymotrypsin-like serine protease domain in the heavy chain. FVIIa requires tissue factor (TF), a membrane bound protein, as an essential cofactor for maximal activity towards its biological substrates Factor X, Factor IX and Factor VII (FVII). Inhibition of TF.FVIIa activity may prevent the formation of fibrin clots and thus be useful in the management of thrombotic disease. The development of TF.FVIIa inhibitors to validate this target has been of great interest. A wide array of strategic approaches to inhibiting the biochemical and biological functions of the TF.FVIIa complex has been pursued. This has been greatly aided from our understanding of the structures for TF, FVII, FVIIa, and the TF.FVIIa complex. These approaches have resulted in inhibitors directed specifically towards either FVIIa or TF. Antagonists include active site inhibited FVIIa, TF mutants, anti-TF antibodies, anti-FVII/FVIIa antibodies, naturally-occurring protein inhibitors, peptide

exosite inhibitors, and protein and small molecule active site inhibitors. These antagonists can inhibit catalysis directly at the active site as well as impair function by binding to exosites that may interfere with substrate, membrane, or cofactor binding. The rationale of TF.FVIIa as a target and the development, characteristics and biological uses of TF.FVIIa inhibitors are discussed.

ACCESSION NUMBER: 2004475345 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15379712
TITLE: Inhibitors of Tissue Factor.Factor VIIa for anticoagulant therapy.
AUTHOR: Lazarus Robert A; Olivero Alan G; Eigenbrot Charles; Kirchhofer Daniel
CORPORATE SOURCE: Departments of Protein Engineering, Genentech, Inc., 1 DNA Way, South San Francisco, CA 94080, USA.. lazarus.bob@gene.com
SOURCE: Current medicinal chemistry, (2004 Sep) Vol. 11, No. 17, pp. 2275-90. Ref: 66
Journal code: 9440157. ISSN: 0929-8673.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200502
ENTRY DATE: Entered STN: 25 Sep 2004
Last Updated on STN: 23 Feb 2005
Entered Medline: 22 Feb 2005

L2 ANSWER 3 OF 60 MEDLINE on STN
TI Role of the Gla and first epidermal growth factor-like domains of factor X in the prothrombinase and tissue factor-factor VIIa complexes.
AB Factor X (FX) has high structure homology with other proteins of blood coagulation such as factor IX (FIX) and factor VII (FVII). These proteins present at their amino-terminal extremity a gamma-carboxyglutamic acid containing domain (Gla domain), followed by two epidermal growth factor-like (EGF1 and EGF2) domains, an activation peptide, and a serine protease domain. After vascular damage, the tissue factor-FVIIa (TF-FVIIa) complex activates both FX and FIX. FXa interacts stoichiometrically with tissue pathway inhibitor (TFPI), regulating TF-FVIIa activity by forming the TF-FVIIa-TFPI-FXa quaternary complex. Conversely, FXa boosts coagulation by its association with its cofactor, factor Va (FVa). To investigate the contribution of the Gla and EGF1 domains of FX in these complexes, FX chimeras were produced in which FIX Gla and EGF1 domains substituted the corresponding domains of FX. The affinity of the two chimeras, FX/FIX(Gla) and FX/FIX(EGF1), for the TF-FVIIa complex was markedly reduced compared with that of wild-type FX (wt-FX) independently of the presence of phospholipids. Furthermore, the association rate constants of preformed FX/FIX(Gla)-TFPI and FX/FIX(EGF1)-TFPI complexes with TF-FVIIa were, respectively, 10- and 5-fold slower than that of wt-FXa-TFPI complex. Finally, the apparent affinity of FVa was 2-fold higher for the chimeras than for wt-FX in the presence of phospholipids and equal in their absence. These data demonstrate that FX Gla and EGF1 domains contain residues, which interact with TF-FVIIa exosites contributing to the formation of the TF-FVIIa-FX and TF-FVIIa-TFPI-FXa complexes. On the opposite, FXa Gla and EGF1 domains are not directly involved in FVa binding.

ACCESSION NUMBER: 2003125801 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12529356
TITLE: Role of the Gla and first epidermal growth factor-like domains of factor X in the prothrombinase and tissue factor-factor VIIa complexes.
AUTHOR: Thiec Fabrice; Cherel Ghislaine; Christophe Olivier D

CORPORATE SOURCE: INSERM U143, Hopital Bicetre, 94276 Le Kremlin-Bicetre Cedex, France.
SOURCE: The Journal of biological chemistry, (2003 Mar 21) Vol. 278, No. 12, pp. 10393-9. Electronic Publication: 2003-01-15.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200305
ENTRY DATE: Entered STN: 18 Mar 2003
Last Updated on STN: 6 May 2003
Entered Medline: 5 May 2003

L2 ANSWER 4 OF 60 MEDLINE on STN
TI The factor IX gamma-carboxyglutamic acid (Gla) domain is involved in interactions between factor IX and factor XIa.
AB During hemostasis, factor IX is activated to factor IXabeta by factor VIIa and factor XIa. The glutamic acid-rich gamma-carboxyglutamic acid (Gla) domain of factor IX is involved in phospholipid binding and is required for activation by factor VIIa. In contrast, activation by factor XIa is not phospholipid-dependent, raising questions about the importance of the Gla for this reaction. We examined binding of factors IX and IXabeta to factor XIa by surface plasmon resonance. Plasma factors IX and IXabeta bind to factor XIa with K(d) values of 120 +/- 11 nm and 110 +/- 8 nm, respectively. Recombinant factor IX bound to factor XIa with a K(d) of 107 nm, whereas factor IX with a factor VII Gla domain (rFIX/VII-Gla) and factor IX expressed in the presence of warfarin (rFIX-desgamma) did not bind. An anti-factor IX Gla monoclonal antibody was a potent inhibitor of factor IX binding to factor XIa (K(i) 34 nm) and activation by factor XIa (K(i) 33 nm). In activated partial thromboplastin time clotting assays, the specific activities of plasma and recombinant factor IX were comparable (200 and 150 units/mg), whereas rFIX/VII-Gla activity was low (<2 units/mg). In contrast, recombinant factor IXabeta and activated rFIX/VIIa-Gla had similar activities (80 and 60% of plasma factor IXabeta), indicating that both proteases activate factor X and that the poor activity of zymogen rFIX/VII-Gla was caused by a specific defect in activation by factor XIa. The data demonstrate that factor XIa binds with comparable affinity to factors IX and IXabeta and that the interactions are dependent on the factor IX Gla domain.
ACCESSION NUMBER: 2003099832 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12496253
TITLE: The factor IX gamma-carboxyglutamic acid (Gla) domain is involved in interactions between factor IX and factor XIa.
AUTHOR: Aktimur Aysar; Gabriel Melanie A; Gailani David; Toomey John R
CORPORATE SOURCE: Department of Pathology, Vanderbilt University, Nashville, Tennessee 37232, USA.
CONTRACT NUMBER: HL58837 (NHLBI)
SOURCE: The Journal of biological chemistry, (2003 Mar 7) Vol. 278, No. 10, pp. 7981-7. Electronic Publication: 2002-12-20.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200304
ENTRY DATE: Entered STN: 4 Mar 2003
Last Updated on STN: 24 Apr 2003
Entered Medline: 23 Apr 2003

L2 ANSWER 5 OF 60 MEDLINE on STN
TI Factor VII mutant V154G models a zymogen-like form of factor VIIa.
AB Proteolytic cleavage of the peptide bond between Arg(152) and Ile(153) converts the procoagulant protein Factor VII (FVII) to an activated two-chain form (FVIIa). The formation of a salt bridge between Ile(153) and Asp(343) drives the conversion of FVIIa from being zymogen-like to the active form. In the present paper, we describe the novel FVII mutant V154G (Val(154)-->Gly mutation; residue 17 in the chymotrypsin numbering system), found in three FVII-deficient patients, which models a zymogen-like form of FVIIa. Recombinant V154G FVIIa, although normally cleaved, shows markedly reduced activity towards peptidyl substrate and undetectable activity towards macromolecular substrates. Susceptibility of Ile(153) to chemical modification, in either the presence or the absence of tissue factor (TF), suggests that the reduced V154G FVIIa activity is caused by impaired salt-bridge formation, thus resulting in a zymogen-like FVIIa form. The TF-mediated protection from chemical modification of V154A indicated that Gly(154) is responsible for this peculiar feature, and suggests that this region, proximal to the heavy chain N-terminus, is directly involved in the conversion of FVII into FVIIa. V154G FVII was exploited to study the FVII-TF interaction, together with three additional FVII variants that were expressed to serve as models for different FVII forms. The comparison of binding affinities of full-length TF after relipidation in L-alpha-phosphatidylcholine for the zymogen FVII (Arg(152)-->Gln, K (d)=1.04+/-0.27 nM), inactive FVIIa (Ser(344)-->Ala, K (d)=0.27+/-0.06 nM) and a zymogen-like FVIIa (V154G, K (d)=1.15+/-0.16 nM) supports the hypothesis that preferential binding of TF to active FVIIa is insufficient to drive the 10(5)-fold enhancement of FVIIa activity. In addition, the inability of V154G FVIIa to accommodate an inhibitor in the active site, indicating an improperly shaped specificity pocket, would explain the low activity of the zymogen-like form of FVIIa, which is predominant in the absence of TF.
ACCESSION NUMBER: 2003030601 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12358603
TITLE: Factor VII mutant V154G models a zymogen-like form of factor VIIa.
AUTHOR: Toso Raffaella; Bernardi Francesco; Tidd Theresa; Pinotti Mirko; Camire Rodney M; Marchetti Giovanna; High Katherine A; Pollak Eleanor S
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, University of Ferrara, Via Luigi Borsari, 46 Ferrara 44100, Italy.. r-toso@hotmail.com
CONTRACT NUMBER: HL-K0803661 (NHLBI)
SOURCE: The Biochemical journal, (2003 Feb 1) Vol. 369, No. Pt 3, pp. 563-71.
Journal code: 2984726R. ISSN: 0264-6021.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200303
ENTRY DATE: Entered STN: 23 Jan 2003
Last Updated on STN: 19 Mar 2003
Entered Medline: 18 Mar 2003

L2 ANSWER 6 OF 60 MEDLINE on STN
TI Tissue factor - a therapeutic target for thrombotic disorders.
AB Exposure of blood to tissue factor (TF) sets off the coagulation cascade. TF is a transmembrane protein that serves as an essential cofactor for activated coagulation factor VII (FVIIa). TF may be exposed locally by vascular injury (such as balloon angioplasty) or by spontaneous rupture of an atherosclerotic plaque. Expression of TF may

also be induced on monocytes and endothelial cells in conditions like sepsis and cancer, causing a more generalised activation of clotting. TF may thus play a central role in thrombosis in a number of settings, and attention has turned to blocking TF as a means to prevent thrombosis. Inhibiting the inducible expression of TF by monocytes can be achieved by 'deactivating' cytokines, such as interleukin (IL)-4, -10 and -13, or by certain prostanoids; by drugs that modify signal transduction, such as pentoxifylline, retinoic acid or vitamin D(3), or by antisense oligonucleotides. Such approaches are for the most part at a preclinical stage. The function of TF can be blocked by antibodies that prevent the binding of FVIIa to TF; by active site-inhibited FVIIa, which competes with native FVIIa for binding; by antibodies or small molecules that block the function of the TF/FVIIa complex; and by molecules, such as TF pathway inhibitor or nematode anticoagulant peptide C2, which inhibit the active site of FVIIa in the TF/FVIIa complex after first binding to activated factor X. The latter two agents have entered Phase II clinical trials. Perhaps most intriguing is the use of anti-TF agents locally, which holds the promise of stopping thrombosis at a specific site of injury without the bleeding risk associated with systemic anticoagulation.

ACCESSION NUMBER: 2002464222 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12223078
TITLE: Tissue factor - a therapeutic target for thrombotic disorders.
AUTHOR: Houston Donald S
CORPORATE SOURCE: Section of Hematology/Oncology, Department of Internal Medicine, University of Manitoba, 675 McDermot Avenue, Winnipeg, Manitoba, R3E 0V9, Canada..
houston@cc.umanitoba.ca
SOURCE: Expert opinion on therapeutic targets, (2002 Apr) Vol. 6, No. 2, pp. 159-74. Ref: 154
Journal code: 101127833. E-ISSN: 1744-7631.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200509
ENTRY DATE: Entered STN: 12 Sep 2002
Last Updated on STN: 13 Dec 2002
Entered Medline: 12 Sep 2005

L2 ANSWER 7 OF 60 MEDLINE on STN
TI Model of a ternary complex between activated factor VII , tissue factor and factor IX.
AB Upon binding to tissue factor, FVIIa triggers coagulation by activating vitamin K-dependent zymogens, factor IX (FIX) and factor X (FX). To understand recognition mechanisms in the initiation step of the coagulation cascade, we present a three-dimensional model of the ternary complex between FVIIa:TF:FIX. This model was built using a full-space search algorithm in combination with computational graphics. With the known crystallographic complex FVIIa:TF kept fixed, the FIX docking was performed first with FIX Gla-EGF1 domains, followed by the FIX protease/EGF2 domains. Because the FIXa crystal structure lacks electron density for the Gla domain, we constructed a chimeric FIX molecule that contains the Gla-EGF1 domains of FVIIa and the EGF2-protease domains of FIXa. The FVIIa:TF:FIX complex has been extensively challenged against experimental data including site-directed mutagenesis, inhibitory peptide data, haemophilia B database mutations, inhibitor antibodies and a novel exosite binding inhibitor peptide . This FVIIa:TF:FIX complex provides a powerful tool to study the regulation of FVIIa production and presents new avenues for developing therapeutic inhibitory compounds of FVIIa:TF:substrate complex.

ACCESSION NUMBER: 2002403316 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12152682
TITLE: Model of a ternary complex between activated factor VII, tissue factor and factor IX.
AUTHOR: Chen Shu-wen W; Pellequer Jean-Luc; Schved Jean-Francois; Giansily-Blaizot Muriel
CORPORATE SOURCE: CEA Valrho-Site de Marcoule, DSV/DIEP/SBTN, Bagnols-sur-Ceze, France.
CONTRACT NUMBER: HL07695 (NHLBI)
SOURCE: Thrombosis and haemostasis, (2002 Jul) Vol. 88, No. 1, pp. 74-82.
Journal code: 7608063. ISSN: 0340-6245.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200308
ENTRY DATE: Entered STN: 3 Aug 2002
Last Updated on STN: 12 Dec 2002
Entered Medline: 18 Aug 2003

L2 ANSWER 8 OF 60 MEDLINE on STN
TI Predicted solution structure of zymogen human coagulation FVII.
AB A model solution structure for the complete tissue factor-free calcium ion-bound human zymogen FVII (residues 1-406) (FVII) has been constructed to study possible conformational changes associated with the activation process and tissue factor (TF) binding. The initial structure for the present model was constructed using the X-ray crystallographic structure of human coagulation FVIIa/TF complex bound with calcium ions (Banner et al., Nature 1996, 380, 41-46). This model was subsequently subjected to lengthy molecular dynamics simulations. The Amber force field in conjunction with the PME electrostatic summation method was employed. The estimated TF free solution structure was then compared with the currently available X-ray crystal structures of FVIIa (with or without TF, variable inhibitor bound) to estimate the restructuring of FVII due to TF binding and activation. The solution structure of the zymogen FVII in the absence of TF is predicted to be an extended domain structure similar to that of the TF-bound X-ray crystal structure. An additional extension of the serine protease (SP) domain of the zymogen above a reference lipid surface by approximately 7 A was in agreement with experiment. Significant Gla-EGF1 and EGF1-EGF2 interdomain motions in the zymogen were observed. Carbohydrate dimers attached to Ser-52 and Ser-60 did not cause restructuring in this domain. Minimal restructuring of the SP domain is found upon inference of the zymogen from the activated form. The catalytic triad residues maintain the H-bonded network while Lys-341 occupies the S1 specific site in the zymogen.
ACCESSION NUMBER: 2002180461 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11913388
TITLE: Predicted solution structure of zymogen human coagulation FVII.
AUTHOR: Perera Lalith; Darden Thomas A; Pedersen Lee G
CORPORATE SOURCE: Department of Chemistry, University of North Carolina, Chapel Hill 27599-3290, USA.
CONTRACT NUMBER: HL-06350 (NHLBI)
SOURCE: Journal of computational chemistry, (2002 Jan 15) Vol. 23, No. 1, pp. 35-47.
Journal code: 9878362. ISSN: 0192-8651.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200205
ENTRY DATE: Entered STN: 1 Apr 2002

Last Updated on STN: 28 May 2002
Entered Medline: 22 May 2002

L2 ANSWER 9 OF 60 MEDLINE on STN
TI Residues Y179 and H101 of a hydrophobic patch of factor VII are involved in activation by factor Xa.
AB We studied factor Xa activation of human factor VII in hopes of identifying factor VII residues, not adjacent to the cleavage site, involved in this interaction. We made eight factor VIIs with single mutations (N100A, H101A, D102Q, L144A, R147A, Y179A, D186A, and F256A) and two factor VIIs with multiple mutations [MM3 (L144A/R147A/D186A) and MM4 (N100A/H101A/Y179A/F256A)]. Residues in MM3 have previously been identified as affecting factor X activation, and the residues of MM4 are located at a hydrophobic patch of factor VII on the opposite side of the catalytic domain from those in MM3. Only H101A, Y179A, and MM4 were activated significantly more slowly than the wild type. Results of our kinetic analyses showed that the catalytic efficiency of factor Xa for activation of factor VII was 176- and 234-fold higher than that for H101A andY179A, respectively. All the mutants with measurable activity had affinities for tissue factor similar to those of the wild type. The activated hydrophobic patch residues, except N100A, which is adjacent to one of the catalytic residues, had normal activities toward both a small peptide substrate and factor X. The rest of the activated mutants (except D102Q with no activity) had reduced activities toward the small substrate (except R147A) and factor X. We conclude that factor VII activation by factor Xa and factor VIIa's catalytic interaction with factor X involve different regions in the catalytic domain, and residues H101 and Y179, part of an aromatic hydrophobic patch, are specifically involved in factor Xa activation of factor VII.
ACCESSION NUMBER: 2001512854 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11560488
TITLE: Residues Y179 and H101 of a hydrophobic patch of factor VII are involved in activation by factor Xa.
AUTHOR: Jin J; Chang J; Stafford D W; Straight D L
CORPORATE SOURCE: Department of Biology, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina 27599, USA.
CONTRACT NUMBER: HL 38973 (NHLBI)
SOURCE: Biochemistry, (2001 Sep 25) Vol. 40, No. 38, pp. 11405-10. Journal code: 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200110
ENTRY DATE: Entered STN: 19 Sep 2001
Last Updated on STN: 29 Oct 2001
Entered Medline: 25 Oct 2001

L2 ANSWER 10 OF 60 MEDLINE on STN
TI The factor VII zymogen structure reveals reregistration of beta strands during activation.
AB BACKGROUND: Coagulation factor VIIa (FVIIa) contains a Trypsin-like serine protease domain and initiates the cascade of proteolytic events leading to Thrombin activation and blood clot formation. Vascular injury allows formation of the complex between circulating FVIIa and its cell surface bound obligate cofactor, Tissue Factor (TF). Circulating FVIIa is nominally activated but retains zymogen-like character and requires TF in order to complete the zymogen-to-enzyme transition. The manner in which TF exerts this effect is unclear. The structure of TF/FVIIa is known. Knowledge of the zymogen

structure is helpful for understanding the activation transition in this system. RESULTS: The 2 A resolution crystal structure of a zymogen form of FVII comprising the EGF2 and protease domains is revealed in a complex with the exosite binding inhibitory peptide A-183 and a vacant active site. The activation domain, which includes the N terminus, differs in ways beyond those that are expected for zymogens in the Trypsin family. There are large differences in the TF binding region. An unprecedented 3 residue shift in registration between beta strands B2 and A2 in the C-terminal beta barrel and hydrogen bonds involving Glu154 provide new insight into conformational changes accompanying zymogen activation, TF binding, and enzymatic competence. CONCLUSIONS: TF-mediated allosteric control of the activity of FVIIa can be rationalized. The reregistering beta strand connects the TF binding region and the N-terminal region. The zymogen registration allows H bonds that prevent the N terminus from attaining a key salt bridge with the active site. TF binding may influence an equilibrium by selecting the enzymatically competent registration.

ACCESSION NUMBER: 2001418440 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11470437
TITLE: The factor VII zymogen structure reveals reregistration of beta strands during activation.
AUTHOR: Eigenbrot C; Kirchhofer D; Dennis M S; Santell L; Lazarus R A; Stamos J; Ultsch M H
CORPORATE SOURCE: Department of Protein Engineering and, Genentech, Inc., South, San Francisco, CA, USA.. eigenbrot.c@gene.com
SOURCE: Structure (Cambridge, Mass. : 2001), (2001 Jul 3) Vol. 9, No. 7, pp. 627-36.
Journal code: 101087697. ISSN: 0969-2126.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: PDB-1DAN; PDB-1JBU
ENTRY MONTH: 200110
ENTRY DATE: Entered STN: 8 Oct 2001
Last Updated on STN: 8 Oct 2001
Entered Medline: 4 Oct 2001

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 NEWS  7  MAY 19   Derwent World Patents Index to be reloaded and enhanced
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 NEWS  9  MAY 30   The F-Term thesaurus is now available in CA/CAplus
 NEWS 10  JUN 02   The first reclassification of IPC codes now complete in
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 NEWS 11  JUN 26   TULSA/TULSA2 reloaded and enhanced with new search and
                   and display fields
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               AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006.

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FILE 'BIOSIS' ENTERED AT 10:50:25 ON 24 JUL 2006
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=> s peptide and binding and factor Vii
L1          135 PEPTIDE AND BINDING AND FACTOR VII

=> s l1 and factor VIIa
L2           60 L1 AND FACTOR VIIA

=> e dennis/au
E1            1     DENNINSTON J C/AU
E2            1     DENNION G R/AU
E3            0 --> DENNIS/AU
E4           27     DENNIS A/AU
E5           14     DENNIS A B/AU
E6            8     DENNIS A C/AU
E7            2     DENNIS A D/AU
E8            2     DENNIS A E/AU
E9            2     DENNIS A F/AU
E10          36     DENNIS A J/AU
E11           7     DENNIS A J JR/AU
E12           9     DENNIS A L/AU

=> e dennis, m/au
E1            1     DENNIS Y M L/AU
E2            1     DENNIS Z G/AU
E3            0 --> DENNIS, M/AU
E4            1     DENNISEN J F/AU
E5            1     DENNISEN JON F/AU
E6            1     DENNISENKO MIKHAIL/AU
E7            7     DENNISH G/AU
E8            1     DENNISH G 3RD/AU
E9           18     DENNISH G W/AU
E10           2     DENNISH G W 3RD/AU
E11           2     DENNISH G W III/AU
E12           4     DENNISH GEORGE/AU

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     FILE 'MEDLINE, BIOSIS' ENTERED AT 10:50:25 ON 24 JUL 2006
L1          135 S PEPTIDE AND BINDING AND FACTOR VII
L2           60 S L1 AND FACTOR VIIA
                E DENNIS/AU
                E DENNIS, M/AU

=> d l2 ti abs ibib 1-10

L2   ANSWER 1 OF 60     MEDLINE on STN
TI   Factor VIIa stimulates endothelin-1 synthesis in
     TNF-primed endothelial cells by activation of protease-activated receptor
     2.
AB   The mechanisms linking prothrombotic changes to endothelial dysfunction
     and accelerated atheroma formation have yet to be fully defined.
     Expression of TF (tissue factor) on the endothelium is potentially an
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initiating event as binding and activation of FVII (factor VII) can result in thrombosis. Although PAR2 (protease-activated receptor-2) is expressed on vascular endothelium, its precise physiological significance and mechanism of activation have yet to be defined. In the present study, we investigated whether PAR2 can be activated by FVIIa (activated FVII) and induce ET-1 (endothelin-1) synthesis. In bovine aortic endothelial cells pretreated with TNF (tumour necrosis factor-alpha) to increase TF expression, FVIIa stimulated ET-1 synthesis via activation of PAR2. Although FX (factor X) alone was inactive, this response was enhanced by using FVII and FX in combination. Inhibition of the proteolytic activity of FVIIa abolished the response. The PAR2 agonist peptide SLIGKV also enhanced ET-1 release on TNF-pretreated cells. The response to FVIIa was inhibited by a PAR2 antagonist peptide FSLLRY. Inhibition of the p38 MAPK (mitogen-activated protein kinase) reduced PAR2 expression and the ET-1 response. In summary, FVIIa can stimulate ET-1 synthesis in endothelial cells by activating PAR2, demonstrating a potential link between thrombotic processes and endothelial cell dysfunction.

ACCESSION NUMBER: 2005081256 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15548135
TITLE: Factor VIIa stimulates endothelin-1 synthesis in TNF-primed endothelial cells by activation of protease-activated receptor 2.
AUTHOR: Sethi Amarjit S; Lees Delphine M; Douthwaite Julie A; Corder Roger
CORPORATE SOURCE: Department of Experimental Therapeutics, William Harvey Research Institute, Barts & the London School of Medicine & Dentistry, Charterhouse Square, London EC1M 6BQ, UK.. amarjit.sethi@eht.nhs.uk
SOURCE: Clinical science (London, England : 1979), (2005 Mar) Vol. 108, No. 3, pp. 255-63.
Journal code: 7905731. ISSN: 0143-5221.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200504
ENTRY DATE: Entered STN: 16 Feb 2005
Last Updated on STN: 13 Apr 2005
Entered Medline: 12 Apr 2005

L2 ANSWER 2 OF 60 MEDLINE on STN
TI Inhibitors of Tissue Factor.Factor VIIa for anticoagulant therapy.
AB Factor VIIa (FVIIa) is a key serine protease involved in the initiation of the coagulation cascade. It is a glycosylated disulfide-linked heterodimer comprised of an amino-terminal gamma-carboxyglutamic acid-rich (Gla) domain and two epidermal growth factor (EGF)-like domains in the light chain, and a chymotrypsin-like serine protease domain in the heavy chain. FVIIa requires tissue factor (TF), a membrane bound protein, as an essential cofactor for maximal activity towards its biological substrates Factor X, Factor IX and Factor VII (FVII). Inhibition of TF.FVIIa activity may prevent the formation of fibrin clots and thus be useful in the management of thrombotic disease. The development of TF.FVIIa inhibitors to validate this target has been of great interest. A wide array of strategic approaches to inhibiting the biochemical and biological functions of the TF.FVIIa complex has been pursued. This has been greatly aided from our understanding of the structures for TF, FVII, FVIIa, and the TF.FVIIa complex. These approaches have resulted in inhibitors directed specifically towards either FVIIa or TF. Antagonists include active site inhibited FVIIa, TF mutants, anti-TF antibodies, anti-FVII/FVIIa antibodies, naturally-occurring protein inhibitors, peptide

exosite inhibitors, and protein and small molecule active site inhibitors. These antagonists can inhibit catalysis directly at the active site as well as impair function by binding to exosites that may interfere with substrate, membrane, or cofactor binding. The rationale of TF.FVIIa as a target and the development, characteristics and biological uses of TF.FVIIa inhibitors are discussed.

ACCESSION NUMBER: 2004475345 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15379712
TITLE: Inhibitors of Tissue Factor.Factor VIIa for anticoagulant therapy.
AUTHOR: Lazarus Robert A; Olivero Alan G; Eigenbrot Charles; Kirchhofer Daniel
CORPORATE SOURCE: Departments of Protein Engineering, Genentech, Inc., 1 DNA Way, South San Francisco, CA 94080, USA.. lazarus.bob@gene.com
SOURCE: Current medicinal chemistry, (2004 Sep) Vol. 11, No. 17, pp. 2275-90. Ref: 66
Journal code: 9440157. ISSN: 0929-8673.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200502
ENTRY DATE: Entered STN: 25 Sep 2004
Last Updated on STN: 23 Feb 2005
Entered Medline: 22 Feb 2005

L2 ANSWER 3 OF 60 MEDLINE on STN
TI Role of the Gla and first epidermal growth factor-like domains of factor X in the prothrombinase and tissue factor-factor VIIa complexes.
AB Factor X (FX) has high structure homology with other proteins of blood coagulation such as factor IX (FIX) and factor VII (FVII). These proteins present at their amino-terminal extremity a gamma-carboxyglutamic acid containing domain (Gla domain), followed by two epidermal growth factor-like (EGF1 and EGF2) domains, an activation peptide, and a serine protease domain. After vascular damage, the tissue factor-FVIIa (TF-FVIIa) complex activates both FX and FIX. FXa interacts stoichiometrically with tissue pathway inhibitor (TFPI), regulating TF-FVIIa activity by forming the TF-FVIIa-TFPI-FXa quaternary complex. Conversely, FXa boosts coagulation by its association with its cofactor, factor Va (FVa). To investigate the contribution of the Gla and EGF1 domains of FX in these complexes, FX chimeras were produced in which FIX Gla and EGF1 domains substituted the corresponding domains of FX. The affinity of the two chimeras, FX/FIX(Gla) and FX/FIX(EGF1), for the TF-FVIIa complex was markedly reduced compared with that of wild-type-FX (wt-FX) independently of the presence of phospholipids. Furthermore, the association rate constants of preformed FX/FIX(Gla)-TFPI and FX/FIX(EGF1)-TFPI complexes with TF-FVIIa were, respectively, 10- and 5-fold slower than that of wt-FXa-TFPI complex. Finally, the apparent affinity of FVa was 2-fold higher for the chimeras than for wt-FX in the presence of phospholipids and equal in their absence. These data demonstrate that FX Gla and EGF1 domains contain residues, which interact with TF-FVIIa exosites contributing to the formation of the TF-FVIIa-FX and TF-FVIIa-TFPI-FXa complexes. On the opposite, FXa Gla and EGF1 domains are not directly involved in FVa binding.

ACCESSION NUMBER: 2003125801 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12529356
TITLE: Role of the Gla and first epidermal growth factor-like domains of factor X in the prothrombinase and tissue factor-factor VIIa complexes.
AUTHOR: Thiec Fabrice; Cherel Ghislaine; Christophe Olivier D

CORPORATE SOURCE: INSERM U143, Hopital Bicetre, 94276 Le Kremlin-Bicetre Cedex, France.
SOURCE: The Journal of biological chemistry, (2003 Mar 21) Vol. 278, No. 12, pp. 10393-9. Electronic Publication: 2003-01-15.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200305
ENTRY DATE: Entered STN: 18 Mar 2003
Last Updated on STN: 6 May 2003
Entered Medline: 5 May 2003

L2 ANSWER 4 OF 60 MEDLINE on STN
TI The factor IX gamma-carboxyglutamic acid (Gla) domain is involved in interactions between factor IX and factor XIa.
AB During hemostasis, factor IX is activated to factor IXabeta by factor VIIa and factor XIa. The glutamic acid-rich gamma-carboxyglutamic acid (Gla) domain of factor IX is involved in phospholipid binding and is required for activation by factor VIIa. In contrast, activation by factor XIa is not phospholipid-dependent, raising questions about the importance of the Gla for this reaction. We examined binding of factors IX and IXabeta to factor XIa by surface plasmon resonance. Plasma factors IX and IXabeta bind to factor XIa with K(d) values of 120 +/- 11 nm and 110 +/- 8 nm, respectively. Recombinant factor IX bound to factor XIa with a K(d) of 107 nm, whereas factor IX with a factor VII Gla domain (rFIX/VII-Gla) and factor IX expressed in the presence of warfarin (rFIX-desgamma) did not bind. An anti-factor IX Gla monoclonal antibody was a potent inhibitor of factor IX binding to factor XIa (K(i) 34 nm) and activation by factor XIa (K(i) 33 nm). In activated partial thromboplastin time clotting assays, the specific activities of plasma and recombinant factor IX were comparable (200 and 150 units/mg), whereas rFIX/VII-Gla activity was low (<2 units/mg). In contrast, recombinant factor IXabeta and activated rFIX/VIIa-Gla had similar activities (80 and 60% of plasma factor IXabeta), indicating that both proteases activate factor X and that the poor activity of zymogen rFIX/VII-Gla was caused by a specific defect in activation by factor XIa. The data demonstrate that factor XIa binds with comparable affinity to factors IX and IXabeta and that the interactions are dependent on the factor IX Gla domain.
ACCESSION NUMBER: 2003099832 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12496253
TITLE: The factor IX gamma-carboxyglutamic acid (Gla) domain is involved in interactions between factor IX and factor XIa.
AUTHOR: Aktimur Aysar; Gabriel Melanie A; Gailani David; Toomey John R
CORPORATE SOURCE: Department of Pathology, Vanderbilt University, Nashville, Tennessee 37232, USA.
CONTRACT NUMBER: HL58837 (NHLBI)
SOURCE: The Journal of biological chemistry, (2003 Mar 7) Vol. 278, No. 10, pp. 7981-7. Electronic Publication: 2002-12-20.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200304
ENTRY DATE: Entered STN: 4 Mar 2003
Last Updated on STN: 24 Apr 2003
Entered Medline: 23 Apr 2003

L2 ANSWER 5 OF 60 MEDLINE on STN
TI Factor VII mutant V154G models a zymogen-like form of factor VIIa.
AB Proteolytic cleavage of the peptide bond between Arg(152) and Ile(153) converts the procoagulant protein Factor VII (FVII) to an activated two-chain form (FVIIa). The formation of a salt bridge between Ile(153) and Asp(343) drives the conversion of FVIIa from being zymogen-like to the active form. In the present paper, we describe the novel FVII mutant V154G (Val(154)-->Gly mutation; residue 17 in the chymotrypsin numbering system), found in three FVII-deficient patients, which models a zymogen-like form of FVIIa. Recombinant V154G FVIIa, although normally cleaved, shows markedly reduced activity towards peptidyl substrate and undetectable activity towards macromolecular substrates. Susceptibility of Ile(153) to chemical modification, in either the presence or the absence of tissue factor (TF), suggests that the reduced V154G FVIIa activity is caused by impaired salt-bridge formation, thus resulting in a zymogen-like FVIIa form. The TF-mediated protection from chemical modification of V154A indicated that Gly(154) is responsible for this peculiar feature, and suggests that this region, proximal to the heavy chain N-terminus, is directly involved in the conversion of FVII into FVIIa. V154G FVII was exploited to study the FVII-TF interaction, together with three additional FVII variants that were expressed to serve as models for different FVII forms. The comparison of binding affinities of full-length TF after relipidation in L-alpha-phosphatidylcholine for the zymogen FVII (Arg(152)-->Gln, K (d)=1.04+/-0.27 nM), inactive FVIIa (Ser(344)-->Ala, K (d)=0.27+/-0.06 nM) and a zymogen-like FVIIa (V154G, K (d)=1.15+/-0.16 nM) supports the hypothesis that preferential binding of TF to active FVIIa is insufficient to drive the 10(5)-fold enhancement of FVIIa activity. In addition, the inability of V154G FVIIa to accommodate an inhibitor in the active site, indicating an improperly shaped specificity pocket, would explain the low activity of the zymogen-like form of FVIIa, which is predominant in the absence of TF.
ACCESSION NUMBER: 2003030601 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12358603
TITLE: Factor VII mutant V154G models a zymogen-like form of factor VIIa.
AUTHOR: Toso Raffaella; Bernardi Francesco; Tidd Theresa; Pinotti Mirko; Camire Rodney M; Marchetti Giovanna; High Katherine A; Pollak Eleanor S
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, University of Ferrara, Via Luigi Borsari, 46 Ferrara 44100, Italy.. r-toso@hotmail.com
CONTRACT NUMBER: HL-K0803661 (NHLBI)
SOURCE: The Biochemical journal, (2003 Feb 1) Vol. 369, No. Pt 3, pp. 563-71.
Journal code: 2984726R. ISSN: 0264-6021.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200303
ENTRY DATE: Entered STN: 23 Jan 2003
Last Updated on STN: 19 Mar 2003
Entered Medline: 18 Mar 2003

L2 ANSWER 6 OF 60 MEDLINE on STN
TI Tissue factor - a therapeutic target for thrombotic disorders.
AB Exposure of blood to tissue factor (TF) sets off the coagulation cascade. TF is a transmembrane protein that serves as an essential cofactor for activated coagulation factor VII (FVIIa). TF may be exposed locally by vascular injury (such as balloon angioplasty) or by spontaneous rupture of an atherosclerotic plaque. Expression of TF may

also be induced on monocytes and endothelial cells in conditions like sepsis and cancer, causing a more generalised activation of clotting. TF may thus play a central role in thrombosis in a number of settings, and attention has turned to blocking TF as a means to prevent thrombosis. Inhibiting the inducible expression of TF by monocytes can be achieved by 'deactivating' cytokines, such as interleukin (IL)-4, -10 and -13, or by certain prostanoids; by drugs that modify signal transduction, such as pentoxifylline, retinoic acid or vitamin D(3), or by antisense oligonucleotides. Such approaches are for the most part at a preclinical stage. The function of TF can be blocked by antibodies that prevent the binding of FVIIa to TF; by active site-inhibited FVIIa, which competes with native FVIIa for binding; by antibodies or small molecules that block the function of the TF/FVIIa complex; and by molecules, such as TF pathway inhibitor or nematode anticoagulant peptide C2, which inhibit the active site of FVIIa in the TF/FVIIa complex after first binding to activated factor X. The latter two agents have entered Phase II clinical trials. Perhaps most intriguing is the use of anti-TF agents locally, which holds the promise of stopping thrombosis at a specific site of injury without the bleeding risk associated with systemic anticoagulation.

ACCESSION NUMBER: 2002464222 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12223078
TITLE: Tissue factor - a therapeutic target for thrombotic disorders.
AUTHOR: Houston Donald S
CORPORATE SOURCE: Section of Hematology/Oncology, Department of Internal Medicine, University of Manitoba, 675 McDermot Avenue, Winnipeg, Manitoba, R3E 0V9, Canada..
houston@cc.umanitoba.ca
SOURCE: Expert opinion on therapeutic targets, (2002 Apr) Vol. 6, No. 2, pp. 159-74. Ref: 154
Journal code: 101127833. E-ISSN: 1744-7631.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200509
ENTRY DATE: Entered STN: 12 Sep 2002
Last Updated on STN: 13 Dec 2002
Entered Medline: 12 Sep 2005

L2 ANSWER 7 OF 60 MEDLINE on STN
TI Model of a ternary complex between activated factor VII , tissue factor and factor IX.
AB Upon binding to tissue factor, FVIIa triggers coagulation by activating vitamin K-dependent zymogens, factor IX (FIX) and factor X (FX). To understand recognition mechanisms in the initiation step of the coagulation cascade, we present a three-dimensional model of the ternary complex between FVIIa:TF:FIX. This model was built using a full-space search algorithm in combination with computational graphics. With the known crystallographic complex FVIIa:TF kept fixed, the FIX docking was performed first with FIX Gla-EGF1 domains, followed by the FIX protease/EGF2 domains. Because the FIXa crystal structure lacks electron density for the Gla domain, we constructed a chimeric FIX molecule that contains the Gla-EGF1 domains of FVIIa and the EGF2-protease domains of FIXa. The FVIIa:TF:FIX complex has been extensively challenged against experimental data including site-directed mutagenesis, inhibitory peptide data, haemophilia B database mutations, inhibitor antibodies and a novel exosite binding inhibitor peptide . This FVIIa:TF:FIX complex provides a powerful tool to study the regulation of FVIIa production and presents new avenues for developing therapeutic inhibitory compounds of FVIIa:TF:substrate complex.

ACCESSION NUMBER: 2002403316 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12152682
TITLE: Model of a ternary complex between activated factor VII, tissue factor and factor IX.
AUTHOR: Chen Shu-wen W; Pellequer Jean-Luc; Schved Jean-Francois; Giansily-Blaizot Muriel
CORPORATE SOURCE: CEA Valrho-Site de Marcoule, DSV/DIEP/SBTN, Bagnols-sur-Ceze, France.
CONTRACT NUMBER: HL07695 (NHLBI)
SOURCE: Thrombosis and haemostasis, (2002 Jul) Vol. 88, No. 1, pp. 74-82.
Journal code: 7608063. ISSN: 0340-6245.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200308
ENTRY DATE: Entered STN: 3 Aug 2002
Last Updated on STN: 12 Dec 2002
Entered Medline: 18 Aug 2003

L2 ANSWER 8 OF 60 MEDLINE on STN
TI Predicted solution structure of zymogen human coagulation FVII.
AB A model solution structure for the complete tissue factor-free calcium ion-bound human zymogen FVII (residues 1-406) (FVII) has been constructed to study possible conformational changes associated with the activation process and tissue factor (TF) binding. The initial structure for the present model was constructed using the X-ray crystallographic structure of human coagulation FVIIa/TF complex bound with calcium ions (Banner et al., Nature 1996, 380, 41-46). This model was subsequently subjected to lengthy molecular dynamics simulations. The Amber force field in conjunction with the PME electrostatic summation method was employed. The estimated TF free solution structure was then compared with the currently available X-ray crystal structures of FVIIa (with or without TF, variable inhibitor bound) to estimate the restructuring of FVII due to TF binding and activation. The solution structure of the zymogen FVII in the absence of TF is predicted to be an extended domain structure similar to that of the TF-bound X-ray crystal structure. An additional extension of the serine protease (SP) domain of the zymogen above a reference lipid surface by approximately 7 A was in agreement with experiment. Significant Gla-EGF1 and EGF1-EGF2 interdomain motions in the zymogen were observed. Carbohydrate dimers attached to Ser-52 and Ser-60 did not cause restructuring in this domain. Minimal restructuring of the SP domain is found upon inference of the zymogen from the activated form. The catalytic triad residues maintain the H-bonded network while Lys-341 occupies the S1 specific site in the zymogen.
ACCESSION NUMBER: 2002180461 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11913388
TITLE: Predicted solution structure of zymogen human coagulation FVII.
AUTHOR: Perera Lalith; Darden Thomas A; Pedersen Lee G
CORPORATE SOURCE: Department of Chemistry, University of North Carolina, Chapel Hill 27599-3290, USA.
CONTRACT NUMBER: HL-06350 (NHLBI)
SOURCE: Journal of computational chemistry, (2002 Jan 15) Vol. 23, No. 1, pp. 35-47.
Journal code: 9878362. ISSN: 0192-8651.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200205
ENTRY DATE: Entered STN: 1 Apr 2002

Last Updated on STN: 28 May 2002
Entered Medline: 22 May 2002

L2 ANSWER 9 OF 60 MEDLINE on STN
TI Residues Y179 and H101 of a hydrophobic patch of factor VII are involved in activation by factor Xa.
AB We studied factor Xa activation of human factor VII in hopes of identifying factor VII residues, not adjacent to the cleavage site, involved in this interaction. We made eight factor VIIs with single mutations (N100A, H101A, D102Q, L144A, R147A, Y179A, D186A, and F256A) and two factor VIIs with multiple mutations [MM3 (L144A/R147A/D186A) and MM4 (N100A/H101A/Y179A/F256A)]. Residues in MM3 have previously been identified as affecting factor X activation, and the residues of MM4 are located at a hydrophobic patch of factor VII on the opposite side of the catalytic domain from those in MM3. Only H101A, Y179A, and MM4 were activated significantly more slowly than the wild type. Results of our kinetic analyses showed that the catalytic efficiency of factor Xa for activation of factor VII was 176- and 234-fold higher than that for H101A andY179A, respectively. All the mutants with measurable activity had affinities for tissue factor similar to those of the wild type. The activated hydrophobic patch residues, except N100A, which is adjacent to one of the catalytic residues, had normal activities toward both a small peptide substrate and factor X. The rest of the activated mutants (except D102Q with no activity) had reduced activities toward the small substrate (except R147A) and factor X. We conclude that factor VII activation by factor Xa and factor VIIa's catalytic interaction with factor X involve different regions in the catalytic domain, and residues H101 and Y179, part of an aromatic hydrophobic patch, are specifically involved in factor Xa activation of factor VII.
ACCESSION NUMBER: 2001512854 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11560488
TITLE: Residues Y179 and H101 of a hydrophobic patch of factor VII are involved in activation by factor Xa.
AUTHOR: Jin J; Chang J; Stafford D W; Straight D L
CORPORATE SOURCE: Department of Biology, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina 27599, USA.
CONTRACT NUMBER: HL 38973 (NHLBI)
SOURCE: Biochemistry, (2001 Sep 25) Vol. 40, No. 38, pp. 11405-10. Journal code: 0370623. ISSN: 0006-2960.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200110
ENTRY DATE: Entered STN: 19 Sep 2001
Last Updated on STN: 29 Oct 2001
Entered Medline: 25 Oct 2001

L2 ANSWER 10 OF 60 MEDLINE on STN
TI The factor VII zymogen structure reveals reregistration of beta strands during activation.
AB BACKGROUND: Coagulation factor VIIa (FVIIa) contains a Trypsin-like serine protease domain and initiates the cascade of proteolytic events leading to Thrombin activation and blood clot formation. Vascular injury allows formation of the complex between circulating FVIIa and its cell surface bound obligate cofactor, Tissue Factor (TF). Circulating FVIIa is nominally activated but retains zymogen-like character and requires TF in order to complete the zymogen-to-enzyme transition. The manner in which TF exerts this effect is unclear. The structure of TF/FVIIa is known. Knowledge of the zymogen

structure is helpful for understanding the activation transition in this system. RESULTS: The 2 A resolution crystal structure of a zymogen form of FVII comprising the EGF2 and protease domains is revealed in a complex with the exosite binding inhibitory peptide A-183 and a vacant active site. The activation domain, which includes the N terminus, differs in ways beyond those that are expected for zymogens in the Trypsin family. There are large differences in the TF binding region. An unprecedented 3 residue shift in registration between beta strands B2 and A2 in the C-terminal beta barrel and hydrogen bonds involving Glu154 provide new insight into conformational changes accompanying zymogen activation, TF binding, and enzymatic competence. CONCLUSIONS: TF-mediated allosteric control of the activity of FVIIa can be rationalized. The reregistering beta strand connects the TF binding region and the N-terminal region. The zymogen registration allows H bonds that prevent the N terminus from attaining a key salt bridge with the active site. TF binding may influence an equilibrium by selecting the enzymatically competent registration.

ACCESSION NUMBER: 2001418440 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11470437
TITLE: The factor VII zymogen structure reveals reregistration of beta strands during activation.
AUTHOR: Eigenbrot C; Kirchhofer D; Dennis M S; Santell L; Lazarus R A; Stamos J; Ultsch M H
CORPORATE SOURCE: Department of Protein Engineering and, Genentech, Inc., South, San Francisco, CA, USA.. eigenbrot.c@gene.com
SOURCE: Structure (Cambridge, Mass. : 2001), (2001 Jul 3) Vol. 9, No. 7, pp. 627-36.
Journal code: 101087697. ISSN: 0969-2126.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: PDB-1DAN; PDB-1JBU
ENTRY MONTH: 200110
ENTRY DATE: Entered STN: 8 Oct 2001
Last Updated on STN: 8 Oct 2001
Entered Medline: 4 Oct 2001

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=> d his

     (FILE 'HOME' ENTERED AT 10:49:24 ON 24 JUL 2006)

     FILE 'MEDLINE, BIOSIS' ENTERED AT 10:50:25 ON 24 JUL 2006
L1          135 S PEPTIDE AND BINDING AND FACTOR VII
L2           60 S L1 AND FACTOR VIIA
                E DENNIS/AU
                E DENNIS, M/AU

=> s l2 and (cysteine)
L3            0 L2 AND (CYSTEINE)
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